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PO077Evaluating a nurse-led diagnosis pathway in parkinson's disease

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mitochondrial area and count in the sPD cohort ($p<0.05$). The number and area of lysosomes was increased in sPD ($p<0.05$) with 15 sPD patients having a lysosome count >2 SD higher than the average of controls (>154%), but CathepsinD/lysosomal activity was decreased. Treatment with ursodeoxycholic acid (UDCA) improved mitochondrial function, but not lysosomal impairment in sPD patient tissue with combined impairment of mitochondrial and lysosomal function.

Conclusion The detection of distinct pathogenic mechanisms in individual patients with sPD may help for future disease-stratification.

PO077 EVALUATING A NURSE-LED DIAGNOSIS PATHWAY IN PARKINSON'S DISEASE

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Objective To evaluate patient satisfaction with a nurse-led new-diagnosis patient pathway for Parkinson's disease.

Background Receiving a diagnosis of Parkinson's disease can result in patients feeling vulnerable and alone. Our patient-led diagnosis project group has previously identified areas of deficiency and developed and implemented a new nurse-led patient pathway covering the first year post-diagnosis. In this project we aimed to evaluate patient experience of this patient pathway.

Methods A questionnaire was designed to evaluate whether the pathway was meeting objectives, and delivered to 40 patients diagnosed within the new pathway between March and September 2016.

Results 19 responses were received (response rate 38.7%). 100% of patients felt they were fully involved in their treatment decisions; 95% felt that treatment was tailored to their needs; 89% of patients had access to information pre-appointment and knew to bring a partner; 100% of patients were given written information during the appointment; 100% of patients felt supported in their diagnosis; 100% said they were given a point of contact and 88% of patients eligible for clinical research were offered the opportunity.

Conclusion Compared with prior to the new pathway, there is a dramatic increase in patient satisfaction. Cost-effectiveness of the pathway is currently being evaluated.

PO079 CERVICAL DYSTONIA IS ASSOCIATED WITH ABNORMAL REWARD BASED REINFORCEMENT LEARNING

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Background The recent discoveries of genes implicated in striatal dopamine transmission in patients with cervical dystonia have emphasised the central role of the basal ganglia in the pathogenesis of the condition. Dopamine's principle role within the striatum is to bias the action selection function of the basal ganglia towards the best outcome. Our hypothesis was that abnormalities of dopamine neurotransmission would result in a measurable bias in reward based learning in patients with cervical dystonia.

Methods We used a reversal learning task to assess dopamine based learning in a group of 40 patients with cervical dystonia and 40 age matched controls. Exclusion criteria included previous psychiatric diagnosis or current use of psychotropic medication.

Results Patients demonstrated a consistent impairment in reversal learning performance compared to controls (15% less rewards $p<0.02$) but equivalent pre-reversal reward and loss-avoidance performance. The contribution from abnormal prediction error signalling are explored using a combination of computational modelling and fMRI.

Conclusions Patients with cervical dystonia have impaired reward-reversal learning. Treatments which aim to enhance reinforcement learning could be explored as future options for both motor and non-motor symptoms in these patients.

PO080 THE TWO FACES OF A FUNCTIONAL NEUROLOGICAL DISORDER

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Vitamin B12 (Cobalamin) deficiency is a well-known cause of central and peripheral nervous system dysfunction, including sensorimotor peripheral neuropathy. Methylmalonate CoA mutase and homocysteine methyltransferase are cobalamin dependent enzymes. In cobalamin deficiency, the metabolic reactions catalysed by these enzymes are inhibited, resulting in the accumulation of methylmalonic acid (MMA) and homocysteine in the blood. High plasma levels of MMA and homocysteine indicate functional (organic) B12 deficiency in individuals with normal renal function and normal or low B12 level. Nitrous oxide (N_2O) is a poorly recognised cause of vitamin B12 deficiency and subsequent neuropathy/myelopathy. We present a case of 27 year old male who was diagnosed with a functional (psychogenic) right lower limb focal dystonia. His severely painful right leg paroxysmal spasms were treated on more than fifty occasions with Entonox (50:50 n_2O and oxygen mixture) over the five years. He developed an axonal sensorimotor neuropathy and was diagnosed with a functional (organic) B12 deficiency related to N_2O administration. His pre-treatment vitamin B12 levels were normal. However, levels of MMA and homocysteine were high. He was advised complete cessation of N_2O, B12 injections and cognitive behavioural therapy for the functional limb dystonia. Post-treatment, his sensory symptoms resolved and MMA and homocysteine levels normalised.

PO081 MYOCLONUS DYSTONIA AND RUSSELL-SILVER SYNDROME IN A PATIENT WITH A MICRODELETION OF CHROMOSOME 7Q

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